

γ -GCSm (G-4): sc-55585



BACKGROUND

γ -glutamylcysteine synthetase (γ -GCS) is the rate limiting enzyme for glutathione (L- γ -glutamyl-L-cysteinylglycine, GSH) synthesis. GSH is ubiquitous in mammalian cells as a vital intra- and extracellular protective antioxidant. γ -GCS is a heterodimer of a heavy catalytic subunit and a light regulatory subunit that is responsive to inflammation, phenolic antioxidants, heat shock, oxidants and cytokines. The human γ -GCS gene encoding the 367 amino acid catalytic subunit maps to chromosome 6p12. The human γ -GCS gene encoding the regulatory subunit maps to chromosome 1p22-p21. The two subunits of γ -GCS form a heterodimeric zinc metalloprotein that gains activity through formation of a reversible disulfide bond.

REFERENCES

- Sierra-Rivera, E., et al. 1995. Assignment of the gene (GLCLC) that encodes the heavy subunit of γ -glutamylcysteine synthetase to human chromosome 6. *Cytogenet. Cell Genet.* 70: 278-279.
- Anderson, M.E. 1998. Glutathione: an overview of biosynthesis and modulation. *Chem. Biol. Interact.* 111-112: 1-14.
- Rahman, I. 1999. Inflammation and the regulation of glutathione level in lung epithelial cells. *Antioxid. Redox Signal.* 1: 425-447.
- Kondo, T., et al. 1999. Regulation of γ -glutamylcysteine synthetase expression in response to oxidative stress. *Free Radic. Res.* 31: 325-334.
- Rahman, I., et al. 2000. Regulation of redox glutathione levels and gene transcription in lung inflammation: therapeutic approaches. *Free Radic. Biol. Med.* 28: 1405-1420.
- Soltaninassab, S.R., et al. 2000. Multi-faceted regulation of γ -glutamylcysteine synthetase. *J. Cell. Physiol.* 182: 163-170.
- Online Mendelian Inheritance in Man, OMIM™. 2002. Johns Hopkins University, Baltimore, MD. MIM Number: 606857. World Wide Web URL: <http://www.ncbi.nlm.nih.gov/omim/>
- LocusLink Report (LocusID: 2729). <http://www.ncbi.nlm.nih.gov/LocusLink/>

CHROMOSOMAL LOCATION

Genetic locus: GCLM (human) mapping to 1p22.1; Gclm (mouse) mapping to 3 G1.

SOURCE

γ -GCSm (G-4) is a mouse monoclonal antibody raised against amino acids 1-274 representing full length γ -GCSm of human origin.

PRODUCT

Each vial contains 200 μ g IgG_{2b} in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

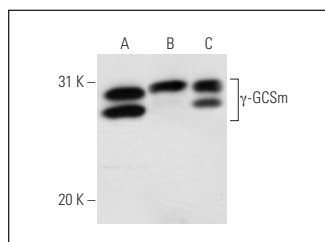
γ -GCSm (G-4) is recommended for detection of γ -GCSm of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for γ -GCSm siRNA (h): sc-40602, γ -GCSm siRNA (m): sc-40603, γ -GCSm shRNA Plasmid (h): sc-40602-SH, γ -GCSm shRNA Plasmid (m): sc-40603-SH, γ -GCSm shRNA (h) Lentiviral Particles: sc-40602-V and γ -GCSm shRNA (m) Lentiviral Particles: sc-40603-V.

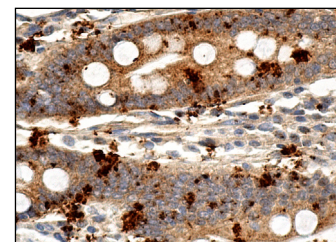
Molecular Weight of γ -GCSm: 31 kDa.

Positive Controls: A549 cell lysate: sc-2413, K-562 nuclear extract: sc-2130 or K-562 whole cell lysate: sc-2203.

DATA



γ -GCSm (G-4): sc-55585. Western blot analysis of γ -GCSm expression in K-562 (A) and A549 (B) whole cell lysates and K-562 nuclear extract (C).



γ -GCSm (G-4): sc-55585. Immunoperoxidase staining of formalin fixed, paraffin-embedded human duodenum tissue showing cytoplasmic staining of glandular cells.

SELECT PRODUCT CITATIONS

- Das Gupta, S., et al. 2015. Dietary γ -tocopherol-rich mixture inhibits estrogen-induced mammary tumorigenesis by modulating estrogen metabolism, antioxidant response, and PPAR γ . *Cancer Prev. Res.* 8: 807-816.
- Zheng, J., et al. 2021. Sorafenib fails to trigger ferroptosis across a wide range of cancer cell lines. *Cell Death Dis.* 12: 698.
- Pontel, L.B., et al. 2022. Acute lymphoblastic leukemia necessitates GSH-dependent ferroptosis defenses to overcome FSP1-epigenetic silencing. *Redox Biol.* 55: 102408.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.